REMARKS

Claims 1, 17, and 23 have been amended to recite that the polypeptide is a factor VIIa polypeptide. No new matter has been introduced. Applicant respectfully requests entry of the above amendments, which raise no new issues that would require further consideration and/or search, and which place the application in better condition for allownace.

Rejection Under 35 U.S.C. § 112, Second Paragraph

The Examiner rejected claims 1, 3-5, 7-14, and 16 under 35 U.S.C. § 112, second paragraph, as being indefinite. Independent claim 1 has been amended to recite that the modified GLA domain comprises an amino acid substitution at amino acid residue 10 and 28. The Examiner is requested to withdraw the rejection under 35 U.S.C. §112, second paragraph.

Double Patenting

The Examiner rejected claims 1, 3-5, 7-14, 16-17, and new claims 23-27 under the judicially created doctrine of obviousness-type double patenting as being unpatentable over claims 1 and 4 of U.S. Patent No. 6,017,882. Applicants will submit a terminal disclaimer upon notification of allowable subject matter.

Rejection under 35 U.S.C. § 102(b)

The Examiner rejected claims 1, 3, 4, 7, 8, 9, 13, and 23-27 under 35 U.S.C. 102(b) as being anticipated by Cheung et al. (Thromb. Res. (1995) 79(2):199-206; Ref. AQ in IDS of Paper No. 24). The Examiner asserted that the Cheung et al. reference discloses a factor VII polypeptide comprising a modified GLA domain comprising a substitution of glutamine at position 10 of SEQ ID No:3 or 4 and a glutamate at position 32 of SEQ ID NO:3 or 4 (see mutant 4 in Fig. 1, page 202)." The Examiner further asserted that although the Cheung et al. reference does not teach that the mutant factor VII has enhanced membrane binding affinity or activity relative to the corresponding native factor VII, it would be inherent that mutant 4 of Cheung et al. would have the claimed functional characteristics.

Independent claims 1 and 23 have been amended to recite that the polypeptide is a factor VIIa polypeptide. Factor VIIa is the active form of factor VII. The Cheung et al. reference does

not disclose such a polypeptide. Rather, the Cheung et al. reference discloses epitope mapping of a monoclonal anitbody using mutant factor VII proteins. Factor VII is the inactive precursor, or zymogen, of factor VIIa and has low enzyme activity. See the present specification at page 1, line 26 through page 2, line 7. Proteolytic cleavage of the zymogen activates factor VII and forms factor VIIa, which in turn, can activate factors IX and X. Factor VIIa differs from factor VII in that it is a two-chain polypeptide and has a different chemical composition due to the addition of water. It also has an altered conformation that allows for enzyme activity. See U.S. Patent No. 5,824,639 (reference AC on the Form 1449 submitted with the Information Disclosure Statement of August 17, 2000). Since the Cheung et al. reference does not disclose or suggest factor VIIa polypeptides, the Cheung et al. reference does not anticipate the claimed invention. In view of the above remarks, the Examiner is requested to withdraw the rejection under 35 U.S.C. §102(b).

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